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TEA DRINKING, PASSIVE SMOKING, SMOKING DECEPTION AND SERUM COTININE IN THE SCOTTISH HEART HEALTH STUDY

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Abstract—Following a recent claim that the use of cotinine in body fluids, to assess passive smoking and smoking "deception", was confounded by metabolic individuality, and by non-tobacco sources of dietary nicotine, particularly tea, data were examined from a large cross-sectional survey in a tea-drinking population. In 3383 men and women aged 40–59 years from the Scottish Heart Health Study, defined as non-smokers, both by self-report and by low thiocyanate and expired air carbon monoxide levels, serum cotinine showed minimal association with self-reported daily average tea consumption. However, there was a strong correlation between degree of self-reported passive tobacco smoke exposure and median serum cotinine level. In the same survey, serum cotinine in 4144 self-reported non-smokers and in 3326 smokers showed entirely different distributions, but the same range, suggesting heavy nicotine intake in some "non-smokers". These analyses confirm that cotinine levels in true non-smokers reflect far more the nicotine in inhaled ambient tobacco smoke than they do nicotine in tea. Some smoking "deceivers" have the same degree of exposure to nicotine as heavy smokers. Despite individual variability, the claim of confounding is poorly supported, and cotinine is confirmed as an indicator both of passive smoking and of smoking deception.

Cotinine Passive smoking Smoking deception Tea drinking Food frequency questionnaire

INTRODUCTION

In a recent review in this **Journal**, Idle [1] questioned the use of cotinine to identify "smoking deceivers", and claimed that its reliability in assessing passive smoking was confounded by nicotine in certain foods, particularly tea. In the accompanying paper [2] we report on smoking habits and smoking biochemistry in 10,359 Scottish men and women in the Scottish Heart Health Study, one of the largest cross-sectional studies to use serum cotinine. We have now used this study to test Idle's suggestion concerning tea, because this population has a high prevalence both of tea drinking, the traditional national beverage, and of passive smoking. In addition, we have looked at the distribution of the range of serum cotinine readings in self-reported smokers and non-smokers to demonstrate "deception".

MATERIALS AND METHODS

The design and conduct of this study are described in the accompanying paper [2]. This was a random cross-sectional sample of 10,359 men and women aged 40–59 years, conducted between 1984 and 1986, in which a cardiovascular questionnaire and examination [3] included questions on food frequency and

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measurements of expired air carbon monoxide (CO), serum thiocyanate and serum cotinine [4]; although technical problems and inadequate serum meant that all three smoking test results were not available for all participants. The questionnaire asked non-smokers whether they had been exposed to tobacco smoke from someone else in the last 3 days, and how much.

Our first analysis concerned passive smoking. We have analysed median serum cotinine levels, for each degree of self-reported passive smoking, against the reported average number of cups of tea consumed daily. The analysis was restricted to those self-reported non-smokers who had an expired air CO level of <6 ppm, and whose serum thiocyanate was <63.4 μmol/l, cut-points identified by us as distinguishing false from true non-smokers [5]. The objective was to remove the effect of any smoking "deceivers" by using cut-points for the two smoke inhalation tests other than cotinine, so that we could observe the uncontaminated effects of passive smoking and tea alone. Median values of cotinine are used, as in the accompanying paper [2], because of the highly skewed distribution.

In the second analysis, to demonstrate differently the existence of smoking "deceivers", we have analysed the distribution of serum cotinine both in self-reported non-smokers (without the above restrictions on thiocyanate and CO) and in those who smoked any tobacco product, using the range, median and 25th and 75th centiles (i.e. the quartile points).

RESULTS

Table 1 shows serum cotinine levels for men and women separately for 4 levels of tobacco smoke exposure and for 5 levels of tea consumption. There is a possible step-up in serum cotinine in those drinking 10 or more cups of tea a day, but the effect is inconstant and there is no consistent gradient below this. Passive smoking however, shows a consistent gradient in median cotinine level which is much more powerful: 9 of the 10 sub-groups of men and women who deny exposure to tobacco smoke have median cotinine levels of 0 or 0.01 ng/ml; whereas 7 out of 10 sub-groups exposed to "a lot" of tobacco smoke had median levels of 1.0 ng/ml or more. Those exposed to "a little" or "some" tobacco smoke were intermediate.

Table 2 shows the range of serum cotinine in those who reported any form of tobacco smoking and in those who denied any such smoking habit. The lowest value of serum cotinine is 0

Table 1. Median serum cotinine levels (ng/ml) by sex, daily consumption of tea and self-reported level of exposure to tobacco smoke in confirmed non-smokers

Exposure to tobacco smoke		Daily cups of tea 0	1–3	4–6	7–9	⩾10	All
"None"							
Men		0.01	0.01	0.01	0.00	0.32	0.01
	n	36	104	132	36	11	319
Women		0.00	0.00	0.01	0.01	0.01	0.01
	n	37	185	204	51	16	493
"A little"							
Men		0.01	0.48	0.26	0.40	0.95	0.40
	n	61	256	259	53	30	659
Women		0.00	0.09	0.01	0.01	0.02	0.01
	n	60	206	220	57	24	567
"Some"							
Men		1.55	1.85	1.81	1.25	1.86	1.81
	n	52	145	151	44	15	407
Women		1.90	0.45	0.82	0.01	0.05	0.59
	n	46	170	205	50	20	491
"A lot"							
Men		1.52	2.25	1.42	0.21	0.73	1.66
	n	11	76	67	18	7	179
Women		2.43	0.77	1.67	1.87	1.23	1.58
	n	23	83	116	32	14	268
"All"							
Men		0.53	0.77	0.46	0.31	0.12	0.56
	n	160	581	609	151	63	1564
Women		0.01	0.01	0.01	0.01	0.05	0.01
	n	166	644	745	190	74	1819

A confirmed non-smoker is a self-reported non-smoker with any cotinine result, serum thiocyanate <63.4 μm/l and CO <6 ppm.

Table 2. Range and quartile points of serum cotinine (n_g/ml) by sex and by self-reported smoking status

Sex	Status	Number	Minimum	25th Centile	Median	75th Centile	Maximum
Male	Non-smoker	1873	00.00	00.00	00.68	3.13	640.00
	Smoker	1940	00.00	123.00	250.00	358.0	998.00
Female	Non-smoker	2271	00.00	00.00	00.10	2.14	932.00
	Smoker	1386	00.00	152.00	243.00	336.00	927.00

A non-smoker is any self-reported non-smoker with a cotinine result. A smoker is any self-reported smoker, of any tobacco product, with a cotinine result.

in both groups and in both sexes. However, the readings in smokers rise rapidly to over 100 ng/ml at the 25th centile, to approx. 250 ng/ml at the median value in both sexes and to nearly 1000 ng/ml at the maximum. For the greater part of the range, in self-reported non-smokers, the cotinine levels reveal no or very little exposure to nicotine. Over a quarter of the population record no measurable levels and the median and 75th centile levels, although measurable, are several hundred times lower than those in the equivalent smokers, and consistent with different degrees of passive smoking shown in Table 1. However, the maximum value, in self-reported non-smokers of both sexes, is of the same order as the maximum in smokers, and many times higher than that of the median self-reported smoker. The number of alleged non-smokers at these levels was too small in percentage terms to show up well in a cumulative frequency distribution graph but numerically significant: there were 95 men and 86 women with serum cotinine levels above 15 ng/ml; 73 men and 68 women above 20 ng/ml; 59 men and 51 women above 30 ng/ml; 49 men and 43 women above 50 ng/ml; and 30 men and 30 women above 100 ng/ml.

DISCUSSION

We are unable to substantiate the claim that tea drinking will confound the effects of passive smoking on serum cotinine. Imperfect though it is, serum cotinine appears to be an indicator of passive smoking. This is shown by the strong gradient in median level, for different levels of reported tobacco smoke exposure, in self-declared non-smokers with low levels of expired air CO and serum thiocyanate. Even the low but measurable cotinine levels in non-smokers who deny exposure to tobacco smoke could, nonetheless, come from tobacco, if they have forgotten their exposure, or because nicotine can be absorbed through the skin, as well as the lungs, from environmental sources.

The range of cotinine readings in Table 2 shows that some self-reported non-smokers are absorbing the same amount of tobacco products as the heaviest smokers: a similar pattern was found for serum thiocyanate and expired air CO. The questionnaire invited respondents both to report whether they smoked, what they smoked, and how much they smoked, and each self-report was checked subsequently at interview, coded, verified and keypunched in duplicate, and also examined for logical inconsistencies on the computer. The discrepancy between self-report and biochemistry is genuine.

We are reporting our criteria and findings for smoking deception using all three biochemical markers in a different analysis elsewhere [5]. Table 2 and the accompanying results show that some self-reported non-smokers are absorbing nicotine at rates that are tens, or even hundreds, of times greater than those attributable to passive smoking, and which must therefore be from deliberate exposure through tobacco smoking.

The discrepancy in numbers between Tables 1 and 2 is accounted for by some missing biochemical values as well as by those non-smokers who had either high thiocyanate or high CO levels. It does not therefore equate exactly with the numbers who exceeded these two cut-points for identifying possible smoking deceivers. The differences from the numbers in the accompanying paper [2] are also explained, either by different smoking categories, or different requirements for levels of completeness of biochemical tests and questionnaire responses.

In our opinion these results support the use of cotinine both to assess passive smoking and to identify "smoking deceivers". "Deceiver" may be a pejorative term. However, it is a problem common to all addictions that become socially unacceptable (alcohol and gambling as well as cigarettes) that, for some individuals, making a false claim to have quit may be easier than quitting. Pharmacological tests are increasingly used to test for furtive drug taking, for example, in athletes. While not denying the possibility of some dietary contribution to serum cotinine, and the variation in individual metabolism

described by Idle [1] we assert nonetheless that passive smoking is the major cause of measurable low levels of cotinine in true non-smokers, and that high levels are virtually pathognomic of furtive smoking, otherwise known as smoking deception. With social pressures to conceal active smoking, and current interest in the harm to health of passive smoking, the value of cotinine measurement should not be underrated.

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